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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

Functional Intestinal Obstruction in an Infant.
Michael A. Brescia, M.D., and Edward F. Tartaglione, M.D. 337

Influenzal Meningitis—Evaluation of Its Treatment. Report of Case with Recovery.
Harry Apfel, M.D. 393

Acute Anterior and Acute Posterior Poliomyelitis. Are They the Same Disease?
Ralph R. Seabay, M.D. 402

Amyotonia Congenita. Report of Two Cases and Discussion.
John P. Albanese, M.D. 411

Pediatrics Half a Century Ago. (Infections of the Newborn).
Samuel McC. Hamill, M.D. and William R. Nicholson, M.D. 419

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(1) Harrell, R.F.: J. Nutrition 31:283 (Mar.) 1946.
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CONTENTS

ORIGINAL COMMUNICATIONS

Functional Intestinal Obstruction in an Infant.
MICHAEL A. BRESCIA, M.D., AND EDWARD F. TARTAGLIONE, M.D. 387

Influenzal Meningitis—Evaluation of Its Treatment. Report of Case with Recovery.
HARRY APFEL, M.D. 393

Acute Anterior and Acute Posterior Poliomyelitis. Are They the Same Disease?
RALPH R. SCOBEY, M.D. 402

CLINICAL REVIEW

Amyotonia Congenita. Report of Two Cases and Discussion.
JOHN P. ALBANESE, M.D. 411

(Continued on page 5)

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(Continued from page 3)

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Infections of the Newborn.
SAMUEL McC. HAMILL, M.D., AND WILLIAM R. NICHOLSON, M.D. 419

BOOK REVIEWS

New Gould Medical Dictionary. Edited by Harold Wellington Jones, M.D., Normand L. Hoerr, M.D., and Arthur Osol, Ph.D. 436

Healthy Babies Are Happy Babies. By Josephine H. Kenyon, M.D., and Ruth K. Russell, M.D. 436

ITEMS

Erythema Nodosum After Administration of Sulfathiazole 392

Lipocaic Insufficiency in Juvenile Diabetes 401

Leukotomy in Children 418

Pneumonia in the Newborn from Inhalation of Gastric Contents 435

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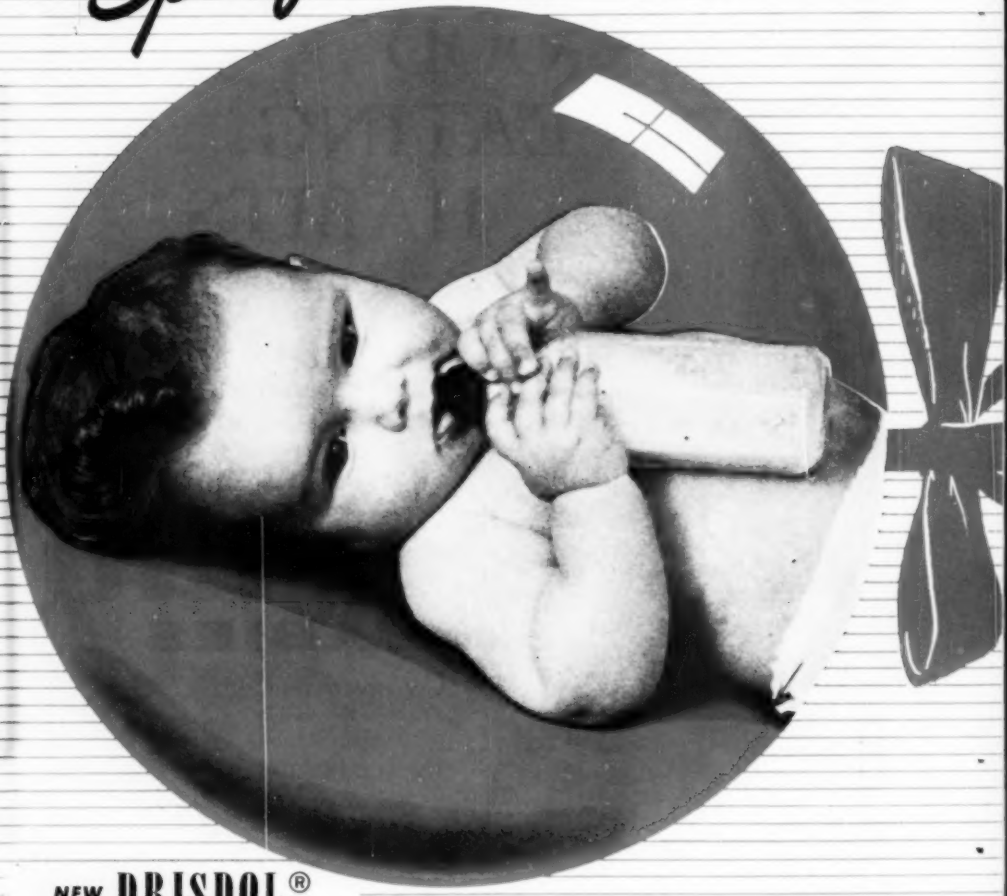

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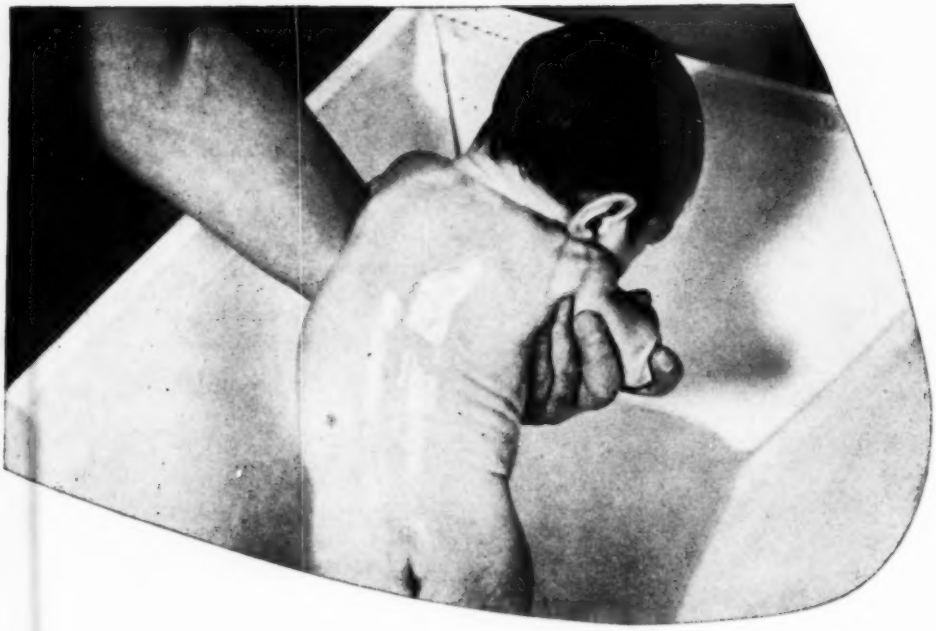

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FUNCTIONAL INTESTINAL OBSTRUCTION IN AN INFANT*

MICHAEL A. BRESCIA, M.D.

AND

EDWARD F. TARTAGLIONE, M.D.

Queens, N. Y.

This case is reported because of the apparently hopeless situation with which we were dealing and the unusual findings which were discovered on x-ray study of the gastro-intestinal tract. This is a case of a newborn male child who vomited from birth and did not have a stool until the fifth day after birth. We thought at first that we were dealing with some type of intestinal obstruction on an anomalous basis. The gastro-intestinal study revealed an obstruction at the cardiac end of the esophagus. In the first few days after birth it could not be decided whether this was an organic obstruction or a functional one.[1] The question of surgical approach to the problem was entertained because of the precarious condition existing but procrastination prevailed and a happy result followed.

CASE REPORT

The mother of the infant was thirty-eight years and the father fifty-six years. Both are well. The mother has a kyphosis. This was the mother's first pregnancy. The last menstrual period was on March 31, 1948 and the expected date of confinement was Janu-

*From St. Johns Long Island City Hospital.

ary 7, 1949. The pregnancy was normal. However, on November 19, 1948 the mother started premature labor and was immediately admitted to St. John's Long Island City Hospital. After a ten-hour labor, the mother was delivered of a normal male infant weighing four pounds twelve and one-half ounces. Outlet forceps were used to facilitate delivery. The infant cried spontaneously and was apparently normal. Soon after delivery the infant vomited some green tinged mucus. This was not thought to be significant. However, the infant would not suck and had to be fed by gavage. During the first twenty-four hours the infant was given water and dextrose by gavage, and promptly vomited all feedings. At this time the abdomen was markedly distended. The infant appeared feeble. Gastric and intestinal peristaltic waves were noted over the entire abdomen. The question of pyloric obstruction was considered even though vomiting due to pyloric stenosis is not expected to appear in the first few days of life. The infant was then placed on an evaporated milk formula (by gavage) with atropine and phenobarbital. The phenobarbital was discontinued after twenty-four hours because the infant seemed to become stuporous. The atropine, however, was continued. The infant continued to vomit all the gavage feedings and no stools were passed. A catheter was inserted in the rectum and left in situ to relieve some of the abdominal distention. The hydration of the infant was maintained by daily hypodermoclysis of normal saline. On the third postnatal day whisky was used to stimulate the infant. The main symptoms at this time were: 1. lack of sucking reflex, 2. frequent bouts of cyanosis and cessation of respiration which necessitated frequent manual resusitation, 3. vomiting all gavage feedings, 4. marked abdominal distention with visible peristaltic waves, 5. absence of stools, and 6. loss of weight. On the fourth postnatal day the infant was subjected to a gastro-intestinal x-ray study. This was accomplished by passing the barium mixture through a Levine tube. The infant managed to vomit part of the mixture while being gavaged but enough remained in the esophagus and stomach to somewhat clarify the picture with which we were dealing. The first films revealed retention of barium in the esophagus. This was so even though the mixture was placed in the stomach as noted by the position of the tube, but by reverse peristalsis the barium was passed back into the esophagus where it was trapped. We could not have a complete obstruction of the

cardiac end of the esophagus since the Levine tube passed readily into the stomach (Fig. 1). Pictures taken two hours later reveal barium still in the lower third of the esophagus assuming a triangular shape with the apex pointing to the obstruction of the cardiac

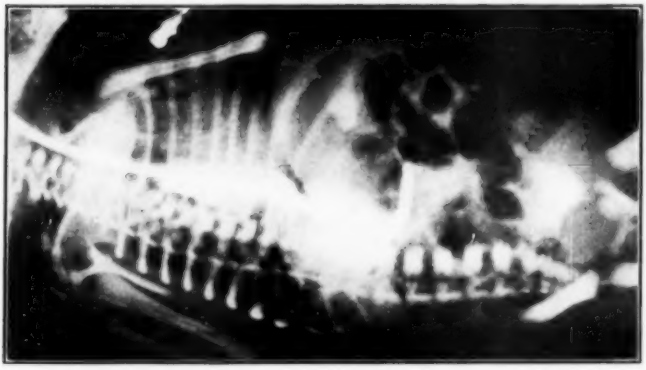

Fig. 1. Right oblique view. The Levine tube is in situ. Barium mixture is seen retained in the lower third of the esophagus.

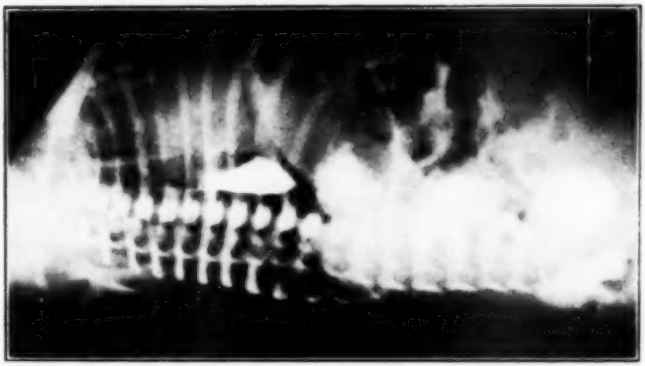

Fig. 2. Right oblique view. Film taken two hours after the one shown in Fig. 1 reveals barium mixture still present in the lower third of the esophagus.

end of the esophagus (Fig. 2). However, whatever obstruction there is there could not be complete since barium is noted in the stomach and also in the small intestines which more or less rules out a pyloric stenosis.

After a study of these pictures it was decided to continue to gavage the infant with an evaporated milk formula and continue with the atropine to the point of flushing. The day following the gastro-intestinal series the infant retained some of its feedings and had for the first time a small (about the size of a peanut) yellow-green stool. The next day the infant retained a little more of its feedings, passed some flatus and had a small green stool. The following three days the infant retained completely three of its feedings. Then on November 29 the infant retained all its feedings for the first time. On December 1 (twelfth postnatal day) the infant for the first time showed strong sucking movements and was fed by nipple. Thereafter the infant took and retained all its feedings and gradually gained weight. On December 13 the

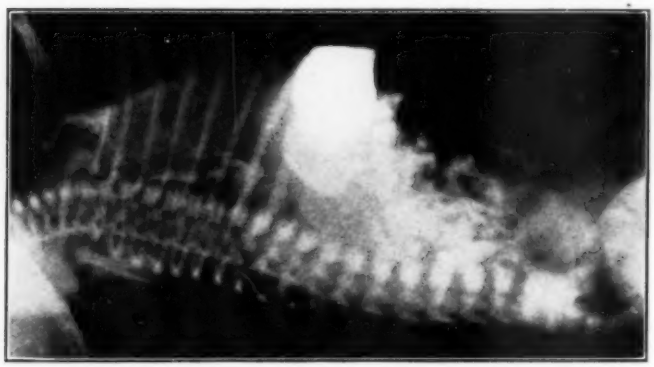

Fig. 3. Right oblique view. No barium mixture retained in the esophagus.

gastro-intestinal series was repeated. This time the infant took the barium mixture by nipple and the series revealed a normal passage of the barium mixture (Fig. 3.). There were no ill effects noted by the use of the barium mixture. Since being home the infant has continued to progress normally and at four months of age is a normal active-appearing infant comparable to an infant of similar age. The weight progress he has attained is demonstrated by the weight curve (Chart 1).

DISCUSSION

It is difficult to explain the pathological physiology involved in this case. The infant behaved as if he had an intestinal obstruction when we consider the cardinal symptoms in this case of vom-

iting, obstipation and visible peristaltic waves over the entire abdomen. Whether or not this was a functional obstruction on a neurogenic basis as described by Zuelzer and Wilson,[1] we cannot state categorically. But nevertheless in a matter of a week the intestinal difficulty adjusted itself and the infant went on to develop

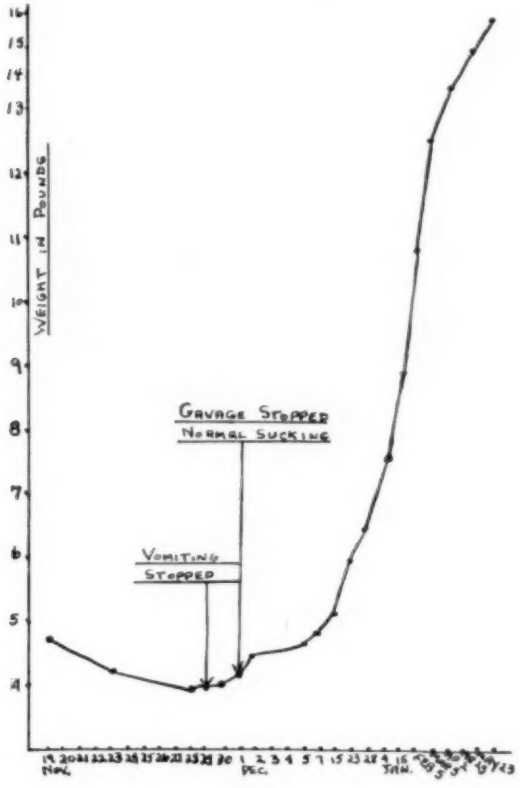

Chart 1.

normally. The temporary obstruction, which was clearly shown by x-ray at the cardiac end of the esophagus, could very well have been due, as Zuelzer and Wilson explained, to deficient functioning of the myenteric plexus, and soon after birth the plexus took on its normal function. Of course the infant was born prematurely,

which may have been a contributing factor to the imperfect functioning of the myenteric plexus. Fortunately this infant was not subjected to surgery although the temptation to do so was great. The mortality rate was very high in those cases subjected to surgery in the series described by Zuelzer and Wilson.

Acknowledgement: We wish to thank Sr. St. Camillus, R.N., and Miss Vera Kozenka, R.N., for their diligent care of this infant without which the infant would not have survived.

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ERYTHEMA NODOSUM AFTER ADMINISTRATION OF SULFATHIAZOLE. (Archives of Disease in Childhood, London, 23: 273, Dec. 1948). According to Doxiadis and McLean the occurrence after the administration of sulfathiazole of a rash similar to erythema nodosum (sulfathiazole erythema nodosum) has been reported frequently. Loefgren has investigated this problem most thoroughly in adults and has concluded that the eruption is not an ordinary drug rash, but is similar in nature to erythema nodosum, the sulfathiazole being a provocative factor only. The present investigation confirms Loefgren's general conclusions. The material for this investigation consists of (a) 17 children in whom a course of sulfathiazole preceded the first appearance of a skin rash similar to erythema nodosum, and (b) 32 children to whom sulfathiazole was given for therapeutic or experimental purposes at varying intervals after an eruption of erythema nodosum. Twelve responded with an eruption of lesions similar to erythema nodosum and 20 did not. In addition the case histories of another 18 children with one or more courses of sulfathiazole for various infections were studied. On the basis of clinical biologic and histologic data the authors conclude that this manifestation is not a "drug rash" due to allergic sensitivity to sulfonamide compound, but is fundamentally the same as erythema nodosum. In order to elicit an eruption of erythema nodosum, sulfathiazole must be given to subjects who are in the active stage of one of the infections commonly associated with spontaneous erythema nodosum. It is, therefore, a provocative and not an etiologic factor.—*Journal A.M.A.*

INFLUENZAL MENINGITIS*
EVALUATION OF ITS TREATMENT

Report of Case with Recovery

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Influenzal meningitis is an acute primary disease of the central nervous system caused by the Hemophilus influenzae. This organism was first described by Pfeiffer[1] in 1892 hence it bears his name. This organism may be found in the healthy human nasopharynx and in other parts of the upper respiratory passages[2] and may assume pathogenicity when body resistance becomes reduced. The organism does not grow in ordinary media but grows quite readily on blood agar; it does not take the gram stain. These characteristics are important in its identification and in its differentiation from other organisms. These bacilli may invade the meninges by way of the blood stream. The incubation period is from two to ten days.[3] Margaret Pittman,[4] of the Rockefeller Institute, has collected some 97 different strains from autopsy material, which may explain in part at least the varied types of degree of clinical pictures in different cases and epidemics. The type that attacks the cerebrospinal system is usually more virile. Some strains may become quite fulminating early in the course of the attack, as has been found recorded in the case history of two brothers, one of whom died within 26 hours and the other in 15 hours.[5] It should be pointed out that the presence of this organism in a given case does not preclude the existence of other and equally dangerous organisms. This organism bears no relation to the virus which is the cause of epidemic influenza.

The disease attacks principally young children; 80 per cent of the cases reported in the literature have been in children under two years of age. It is not usually found in neonatal life. The reason for this, as explained by Fothergill and Wright,[6] is the fact that the newly born infant acquires a sufficient number of antibodies from its mother. Unfortunately these protective antibodies tend soon to disappear leaving the young infant nonimmune. Like with most accepted facts there are also exceptions, as evidenced in one case report in 5-day-old twins.[3]

*Read before the Clinical Society of the Unity Hospital, Brooklyn, New York, April 27, 1949.

History. Meningitis is mentioned in the early writings of Avicenna, who lived in 1000 A.D. It was not, however, until Quincke[7] discovered the method of spinal puncture, in 1891, which made it possible to establish the diagnosis of the type of the disease clinically during the lifetime of the patient. Slawyk[14] is credited with having reported the first case in 1899. The first interest in this disease in our country is credited to a woman pediatrician, Martha Wollstein[8] of the Babies Hospital of New York. It was she who reported 11 cases. This same investigator also is said to have perfected a serum from goats with which she succeeded in curing two monkeys which were first inoculated with the disease.

Prior to 1929 no case was known to have recovered from this disease. In 1930 Jenks and Radhill[9] reported 32 cases without a single recovery. About the same time Rivers[10] reported 23 cases with one recovery. Beck and Jenney[33] cite a series of 66 cases (between 1923-39), with a mortality rate of 95 per cent. In 1940, Silverthorn and Brown[11] reported their experiences with this disease and proudly report a reduced mortality to only 90 per cent. In 1934, Neal, Jackson and Appelbaum[12] collected 111 cases with only four recoveries. Still another investigator, we find in the same period, reports a mortality of 99 per cent.[13]

In 1939, Alexander[15] marks a new epoch in the treatment of this deadly disease with her discovery of the rabbit antibody serum. She obtained a recovery of 35 cases out of a total of 45 so treated. This success was soon repeated by Birdsong[16] and his coworkers, employing the Alexander serum plus sulfadiazine. Then came the present era with its newer antibacterial agents, to wit, penicillin and more recently streptomycin.[18] While the former has no selective action on the Hemophilus influenzae, it nevertheless plays an important part in the treatment of this disease, as we shall show when we discuss the treatment.

Pathology. A critical study of the pathology, together with the clinical course of this disease, has been reported by a Boston group of workers.[17] The material covers a group of 14 cases over a period of 10 years, taken from the files of the Boston City and Massachusetts General Hospital. The period of illness varied from 15 to 76 days. The pathology was found to consist of nerve cell necrosis of the cerebral cortex and thrombosis of the cerebral veins. The convex portion of the brain was found to be covered with

an inflammatory exudate. The ventricles were at times distended and hydrocephalus was frequently found. The smaller vessels showed proliferation of their endothelial and the adventitious cells. The exudate extended to the subarachnoid spaces. The dura mater was coated with a layer of fibrin. The spleen and other viscera showed some degenerativ changes. Otitis media and bronchial pneumonia were frequently found.

Symptomatology and Diagnosis. The classical symptoms of meningitis, of the meningococcic type, are not usually found early in this disease. This is an important factor to be borne in mind; its disregard frequently contributes to the great number of diagnostic failures with the resultant large mortality, even today with our more effective remedies at hand. It behooves us to stress the significance of the less prominent symptoms which, if recognized early, will reward us with a correct diagnosis. Delay of several days may permit the infection to reach the irreparable stage. Vomiting and lethargy are early symptoms. Convulsions may appear at any time within the first two days. They are usually of a generalized nature and tend to be persistent and difficult to control. Fever may be of the low grade. If high, it is usually not of the sustained type but rather intermittant. Intervals of quiescence and freedom from symptoms, except for drowsiness, may be met with. The child may cry out from its sleep as if in pain. Abdominal distention and diarrhea, alternating with constipation, may be present. Nuchal rigidity, hyperesthesia of the abdominal walls, Kernig, Brudzinski signs, etc. are usually absent early in the course of the disease.

Laboratory Findings. The spinal fluid, which is usually found under increased pressure, is cloudy. The cell count is increased into the thousands and of the polymorphonuclear leukocytes. The globulin is increased and the sugar is reduced or entirely absent. The sugar reduction is in direct proportion to the rate of increase in the leukocytes. The direct smear may show the gram-negative, rod-shaped bacilli, which on culture may prove to be the type B Hemophilus influenzae. The blood culture may also be positive. The blood count is of little significance. The urine may show slight traces of albumin and traces of acetone, especially where vomiting was of the persistent type.

Many a young child is the victim of the above enumerated symp-

toms and allowed to continue undiagnosed for several weeks. We came across one such case recently that was allowed to go on untreated for two weeks before the diagnosis was established. Many such cases are signed out as having died of "circulatory failure," etc. We may succeed in reducing these failures and their high mortality by analyzing the "insignificant" symptoms and when in doubt by doing a spinal tap. This will do the child no harm and may clear up the unexplained symptomatology and so hasten the onset of proper treatment.

Treatment. As a guide in the treatment of this disease, we wish to enumerate several basic principles:

1. Regardless of the particular drug or remedy one chooses to employ, it must be instituted as early as possible.

2. Do not procrastinate-waiting for the laboratory report before beginning treatment.

3. In-as-much-as the sulfonamides are the accepted therapeutic agents, the aim should be to establish the optimum blood concentration as early as possible.

4. Do not place all your hopes in any one or two of the recognized remedies. Fidelity in therapeutics is not always a virtue. A former success with a given antibiotic need not deter you from trying out another remedy in your next case. In each instance we may be dealing with a different strain of the organism.

5. Do not be over-optimistic about the intrathecal or the cisternal route. Try first the intramuscular or the intravenous route. Both of these latter routes are simpler and by far safer than the former.

The Antibacterial Agents. Among the several antibacterial remedies available at the present time, the sulfonamides are the most impressive, judging from all reports in the literature. All observers are unanimous in their endorsement of this chemotherapeutic agent as it concerns this disease. The choice of the particular preparation is relatively of little concern. Abroad, sulfamethazine has been employed extensively. In our country, sulfadiazine has become the preparation of choice. The sodium salt of the latter product has become the great favorite of many clinicians, largely because of the encouraging reports by Appelbaum and his coworkers.[17] In our own limited experience, the combination of sulfadiazine and sulfamerazine has given us the best results.

Whichever preparation one decides to employ, the important objective is to obtain a blood concentration of 10-15 mgs.% as early as possible. This will result in a spinal fluid concentration of the strength desired.

The second remedy we wish to mention is the rabbit specific antibody serum, developed by H. E. Alexander.[15] This serum has helped, materially, to reduce the mortality rate of this deadly disease. It should be said, without, prejudice, however, that this remedy is not to be regarded as indispensable. The case reports found in the literature bear out this contention. If, however, you decide to employ the serum, it must be given in from 25-100 mgs. antibody nitrogen, depending on the amount of sugar found in the patient's spinal fluid at the time.

Streptomycin is the newer remedy, discovered by Waksman[18] in 1944, and is, to date, the most important single remedy for the treatment of this disease. It should be given intramuscularly in the dose of 20 mgs. per pound of body weight per twenty-four hours, in divided doses, every three hours.

Penicillin should not be overlooked as an important antibiotic in connection with the above mentioned remedies. While it has no selective action upon the H. influenzae, nevertheless exerts a synergystic influence, according to the best opinion available.[21] This remedy should be given even before the laboratory has confirmed the clinical diagnosis.

Zinnemann[22] of England treated 15 cases with sulfamethazine and penicillin and obtained 8 recoveries. Appelbaum[17] reports 26 cases using sulfadiazine alone, with 65 per cent recoveries. This same author reports a second series of 42 cases, adding the rabbit antiserum with 64 per cent recoveries. These figures in a fairly representative number of cases should convince any skeptic of the value of the chemotherapeutic agent in the treatment of this disease. In nowise should my statement be mistaken as a slur against the other important remedies. Beck and Jenney[23] report their experience with sulfadiazine and serum with a mortality as low as 13.6 per cent. Alexander and Leidy[24] in their more recent article make the following comment which deserves to be quoted in its original text: "In order to combat the sulfa resistant cases, streptomycin should be added; . . . in the streptomycin-fast strains sulfadiazine should be used simultaneously; where those two

remedies fail to give prompt relief, antibody serum must be used."

A Plea Against Intrathecal Therapy. We will commence by making the categorical statement, namely, we are opposed to the intrathecal route in the treatment of any of the acute diseases of the cerebrospinal system. We have come to this conclusion both as a result of the experience of others as well as from personal experience. Walker and his associates,[23] in their experiments with monkeys, have reported urticarias and convulsions following intraspinal injections. Hinshaw and Feldman[24], cite headache and fever as a result of intrathecal therapy. Alexander and her associates[15] cite two cases, showing improvements, which became comatous after two intrathecal injections. One of these two ended fatally. Thomson[25] reports tremors, cyanosis and embarrassed respirations following intrathecal medication. Hoyne and Archibald[26] conclude their experiences on this question as follows: "Good results may be obtained without intrathecal therapy." Drysdale[20] and his collaborators report convulsions in a 20-month-old baby following one intraspinal injection of penicillin. Alexander[27] calls the intrathecal treatment "drastic." Hoyne and Brown,[37] in reporting 28 cases, conclude their experience as follows: "Recovery is more prompt and complications are fewer when the intrathecal treatment is omitted."

One of the reaasons why so many resort to the intrathecal route is largely due to the prevailing impresssion that drugs administered intravenously, or by way of the other parenteral routes, fail to enter the subarachnoid spaces. This contention is not based on fact. Rosenberg and Sylvester,[38] in their studies with patients in the U. S. army, have been able to recover penicillin within 60 minutes after parenteral injection. Cook and Goldring,[28] working with patients in the Children's Hospital of St. Louis, have recovered the drug within 15 minutes after its intramuscular injection. Long[29] has shown that sulfadiazine will find its way into the spinal canal in the amount of ⅔ the quantity found in the blood. Appelbaum[17] has shown that sodium sulfadiazine in a blood concentration of 10-15 mgs.% will give a spinal fluid concentration of 72 per cent of that in the blood at the time. Alexander,[31] after resorting to the intrathecal route extensively, writes as follows: "It is our intent to delay using the intrathecal route until we find the intravenous route will not do;—if the treatment be started early

the intrathecal route is unnecessary." In our own experience over many years with various types of meningitis, we became definitely prejudiced against the intrathecal route[31] for the purpose of medication. According to Faber[36] "the intrathecal route is of little value in acute inflammatory conditions."

CASE REPORT

This child was 7½ months of age and weighed 15 pounds. The previous history was of no consequence. It was admitted to the Unity Hospital on November 3, 1948. The history, obtained from the mother, showed that the child had vomited and appeared drowsy the day before. On the morning of admission the child was seized with a convulsion, involving the entire body, and continued unabated for about 3 hours. The temperature at the time was 104° F. After we succeeded in checking the convulsion the child fell asleep and remained asleep for several hours during which time the temperature dropped to normal. For the remainder of the first hospital day the child took its feedings and retained them. Physical examination on the same day showed no findings of any consequence, except for a mild pharyngitis. We were confronted with the puzzling question, why the severe convulsion? We deferred making a definite diagnosis for the time being. The following morning the nurse's notes reported the child having spent a restless night, crying out at intervals, as if in pain. All this time the temperature and respiration remained normal. The laboratory reported the blood count to be normal, except for slight achromia. The urine showed 1+ albumin. Again the physical examination was quite negative except for drowsiness. We felt that this called for a spinal tap to rule out or to corroborate a working diagnosis of "acute meningitis."

The spinal fluid was found to be under considerable pressure and decidedly cloudy. We removed about 12 cc. The laboratory reported the cell count to be 3,600 per cm. The cells were of the polymorphonuclear leukocyte type. The globulin, 130 mgs.%; sugar, 5 mgs.%. Direct smear showed many gram-negative bacilli. The culture later showed these to be the type B Hemophilus influenzae. Our laboratory dispatched some of this fluid to the New York City Department of Health Laboratory. The report returned to us later corroborated the findings of our hospital laboratory. We were

happy to learn that the strain of the organism we were dealing with was quite sensitive to streptomycin. (It took 1.56 units per cc. to sterilize a culture containing 10,000 organisms.)

Treatment prior to the laboratory report, consisted of sulfadiazine and sulfamerazine, every 4 hours; penicillin every 3 hours, intramuscularly. After the laboratory report we added streptomycin. The latter we administered intramuscularly every 3 hours. The first dose was 100,000 (micrograms), and the subsequent doses were 50,000 micrograms each. We tried to maintain a sulfa concentration in the blood around 12 mgs.%. We did *not* resort to any intrathecal therapy. In all we did three spinal taps. These were done for the purpose of diagnosis, the first time; and later to help check the convulsions by reducing the cerebrospinal pressure.

The clinical course was interesting from the standpoint that the temperature and the blood count were in the normal range for the first two hospital days, except for the brief hours on admission when the child was in convulsions. The outstanding symptoms, in addition to the convulsions, were the drowsiness and the vomiting. The abdomen was constantly distended and diarrhea was frequent. Nuchal rigidity developed late in the course of the attack and that was followed by pronounced opisthotonos. The temperature curve was highly irregular, ranging between normal and 104°F. For a period of several days we were resigned for the worst prognosis.

The child finally made a complete recovery and, after 16 hospital days, was discharged fully well. Were it not for the splendid care this child received at the hands of our nurses and the remarkable attention by our bacteriologist, Miss Josephine Bears, this child might not have recovered.

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LIPOCAIC INSUFFICIENCY IN JUVENILE DIABETES. (Semaine des Hôpitaux de Paris, 25: 730, Mar. 2, 1949). Sendrail and Bazex report 4 boys between the ages of 10 and 19 years and 4 girls between the ages of 2 and 20 years with severe diabetes mellitus of insular type preceding the other symptoms of their syndrome, which consisted of infantilism and nanism, a disturbance of the distribution of fat, hepatomegaly and hyperlipoidemia (Mauriac's syndrome). The pathogenesis was confined to a diabetic lipoidosis. Mauriac's syndrome may be interpreted on the basis of a double functional incompetence of the islands of Langerhans, that concerned with regulation of glycogen storage and regulation of lipids, rather than by assuming a failure of the function of the anterior lobe of the pituitary. The occurrence of lipid hepatitis depends on multiple lipotropic factors, the most active of which is Dragstedt's lipocaic hormone. The lipocaic deficiency may be responsible for the complex of symptoms which characterize the syndrome of Mauriac. Juvenile diabetes appears to combine two groups of manifestations, those due to insulin deficiency and those due to lipocaic deficiency.—*Journal A.M.A.*

ACUTE ANTERIOR AND ACUTE POSTERIOR POLIOMYELITIS. ARE THEY THE SAME DISEASE?

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Garrow[1], 38 years ago, in an article entitled, "Are Polio and Herpes Zoster the Same Disease?" brought out facts that require reconsideration in the light of present day failures to explain many phenomena observed during epidemics of poliomyelitis.

Garrow observed during August and September 1911, when acute anterior poliomyelitis was epidemic in Maryport, England, that there was also prevalent an unusual number of cases of herpes zoster. He felt that there was an etiological connection between the two diseases.

It is the purpose of this report to consider in detail the facts that were pointed out by Garrow and to correlate these facts with observations made by other writers.

ETIOLOGIC AND PATHOLOGIC AFFINITIES OF HERPES ZOSTER AND POLIOMYELITIS

Head and Campbell[2] (1900) were the first to apply the term "acute posterior poliomyelitis" to herpes zoster. They pointed out the analogy of herpes zoster and acute anterior poliomyelitis as follows: "A typical attack of zoster arises without any obvious or central cause, and must be considered a specific disease of the nervous system, for it starts with a prodromal period of varying length, during which the temperature is raised. During this period the patient feels ill and has more or less pain; but it is then impossible to make a diagnosis. If the patient is a child he will be supposed to be sickening for one of the ordinary acute specific diseases. Suddenly, however, after a variable period, the rash comes out and the disease declares itself. The febrile period lasts from three to five days and this may be considered the duration of the acute disease. The rash may appear a few hours after the onset of the disease or it may tarry till the fall of the temperature.

"But if herpes zoster is an acute specific disease of the nervous system we find an exactly analogous disease in acute anterior poliomyelitis. This disease begins with malaise and fever. The temperature may remain raised from three days to a week, and

at a variable time during this febrile period, or it may not be until the fever has subsided, paralysis is noticed.

"On comparing the pathological condition discovered on microscopic examination in the two diseases their likeness becomes still closer." Head and Campbell point out that if for the anterior horn we read posterior root ganglion, and for motor cell, posterior ganglion cell, we would have an exact description of the pathological changes that are present in acute anterior poliomyelitis. "Moreover," they say, "the posterior root ganglion is the exact equivalent of the anterior horn. For whereas on the motor side the cells of the neurones of the lowest level lie in the anterior horn, on the afferent side, they happen to lie collected together outside the spinal cord in the posterior root ganglion. Thus zoster might justly be spoken of as acute posterior poliomyelitis.

"The changes which we have described above in the posterior root ganglion, closely resembling as they do those found in the gray matter of the anterior horn in acute anterior poliomyelitis, are typical of a pathological process in the nervous system that has of late years been too much neglected."

Herpes zoster has been observed to occur with more frequency in males than in females including pregnant women. This observation corresponds with that observed in epidemics of poliomyelitis.

Herpes zoster, as observed by a number of writers, occurs most frequently in the summer and fall months. It is during this season, as is well-known, that epidemics of poliomyelitis occur. Garrow was impressed, as stated above, by the simultaneous occurrence of an epidemic of herpes zoster and of poliomyelitis. Beers[2], in the summer of 1935, had a succession of 12 or more cases of herpes zoster in a very short time. Head and Campbell[3] (1900) observed that epidemics of herpes zoster occur. They point out that in 1897, a large number of cases appeared in England during a long drought that lasted from the end of July until the beginning of November. This observation appears to be significant since epidemics of acute anterior poliomyelitis are frequently noted during droughts.

The etiological factors that have been found to give rise to an attack of herpes zoster are variable and nonspecific. Several writers emphasize a toxin as primarily involved. Robinson and Hirschler[4] state: "It is highly probable that the vast majority of all cases of

herpes of the various types are the result of the action of a toxin."

Herpes zoster may result from chemical poisons, bacterial toxins, traumatism and pressure on the spinal cord. Some cases, in which the cause cannot be discovered, are classified as "idiopathic."

Herpes zoster has been attributed to poisoning by a number of poisons, including arsenic, carbon monoxide, gold, mercury, bismuth, barbiturates, sulfonamides and alcohol. Epidemics of herpes zoster have followed the ingestion of arsenic in beer in England. Levitt and Luppi[5] (1941) described cases of herpes zoster with alcoholic polyneuritis. Caspers[6] (1942) described cases of polyneuritis and herpes zoster as late sequels of acute phenobarbital poisoning.

The toxins resulting from a variety of infections have resulted in the production of herpes zoster. In this connection it is significant to note that some of the early workers investigating poliomyelitis were able to produce the disease by nonspecific bacterial toxins.

Traumatism, as a cause of herpes zoster, has been mentioned in the literature for many years. Mitchell, Moorhouse and Keen[7] observed the association of trauma and herpes zoster during the Civil War. Klauder[8] (1947), who gives a review of this subject, states: "It is not reasonable to ascribe herpes zoster appearing after the aforementioned circumstances to a chance occurrence." Pusey[9] states: "These reflex and mechanical causes of herpes zoster, however, represent a small minority of the cases, and do not weaken the fact that in a large majority of the cases the neuritis which underlies it is of toxic origin."

It appears to be significant that a history of falls or injuries is not infrequently obtained in cases of acute anterior poliomyelitis. Thirteen patients, of 74 reported cases, in an epidemic of poliomyelitis in Jamestown, N. Y. in 1947[10], gave such a history. A fatal case had fallen off a horse eight days before her illness. Another severe case stated that he had been thrown from a horse several times during the summer. Anglin[11] (1948) reported a fatal case of acute bulbospinal poliomyelitis in a patient who had fallen out of bed several times during the previous two weeks. The last time this 11-year-old female fell out of bed was three days prior to admission to the hospital. Following this accident the child appeared drowsy and complained of stiffness of the neck.

Smart[12] (1888), in describing an epidemic of paralysis among soldiers, during the Civil War, says: "Overexertion and heavy burdens on the loins would predispose by repetition as surely as a more pronounced injury inflicted but once." He states that injuries may have determined the localization of the paralysis.

Herpes zoster, in addition to the above causes, has been noted to result from pressure caused by Pott's disease, tumors of the spinal cord, myelitis and subarchnoid hemorrhage.

Herpes zoster has been precipitated by exposure to wet and cold, physical exertion and mental exhaustion and anxiety. It has been observed that similar factors often precede an attack of poliomyelitis. Robinson and Hirschler[4] state that in most cases herpes zoster results from causes acting on the body in general though these precipitating factors may assist.

An attack of herpes zoster commonly begins with fever, malaise, anorexia and gastro-intestinal disturbances. It may be ushered in with upper respiratory symptoms, sore throat, etc. There is hyperesthesia and there may be headache. Within three or four days on the average, the eruption makes its appearance. Children are more prone to suffer from malaise; adults frequently have an initial rigor. The fever usually ranges from 100° to 102° F. Hegier[13] (1901) described sweat disturbances in both anterior and posterior poliomyelitis.

There may be evidence of meningeal irritation in herpes zoster as in cases of poliomyelitis. This fact is evidenced by a positive Kernig's sign, exaggerated knee jerks and a pleocytosis with increased protein in the spinal fluid.

The simultaneous occurrence of herpes zoster and poliomyelitis has been noted (Fromenty[14]). Broadbent[15] (1866) was the first to describe paralysis following an attack of herpes zoster. Taterka and O'Sullivan[16] (1943) reviewed the literature on this subject. They found that the zoster eruption preceded the paralysis in about three-fourths of the cases; it followed the paralysis in one-fourth. The intervals between the eruption of herpes zoster and the appearances of the paralysis, or vice versa, varied from one to two months. The average interval was from a few days to a few weeks.

Taterka and O'Sullivan, in their analysis, showed that 16 per cent of the patients recover with no residual abnormalities; the time required for complete recovery was usually one-half to one

year and in only a few cases was it less than two or three months. Full recovery did not occur in 81.7 per cent of the patients; some atrophy and muscular weakness persisted when treatment was discontinued after a year or more.

Some investigators, in attempting to explain these motor complications in connection with herpes zoster, feel that the disease process in the spinal ganglions spreads to the anterior root. "The pathological process is not necessarily limited to the posterior root ganglion." Wohlwill[17] (1924), who tried to demonstrate the variety of pathological processes, found besides the ganglion the following structures involved: posterior root, cord and its membranes, peripheral nerves, less often the anterior roots and the peripheral sympathetic nerves. In a case reported by Lhermitte and coworkers[18], the autopsy revealed not only involvement of the spinal ganglions, posterior root and peripheral nerves but also the posterior horn, gray substance of the anterior horn and very slightly of the anterolateral columns. Parkinson[19] (1948) points out that in some cases of herpes zoster, in which motor symptoms occur, that the anterior horn cells of the spinal cord are affected in a manner similar to that in poliomyelitis. Osler and McCrae[20], years ago, pointed out that the pathology of herpes zoster is analogous to acute anterior poliomyelitis.

Colliver[21] (1917), and other writers since, pointed out that the posterior horns are affected early in cases of poliomyelitis. Colliver states: "As an indication that the posterior horns are first involved, we have as a most constant symptom of the preparalytic stage the hypersensitivity of the skin. This is so marked as to be painful, causing the child to cry when the skin is touched."

It has been found that herpes zoster, like poliomyelitis, rarely occurs twice in the same individual. Head and Campbell[2] (1900) state regarding this fact: "If zoster be an acute specific disease, second attacks should be uncommon, and we find as a fact that second attacks are less common than second attacks of measles. We have met with only four instances in over four hundred cases."

ATYPICAL FORMS OF HERPES ZOSTER AND POLIOMYELITIS

Several atypical forms of herpes zoster have been described. These include: herpes sine hepete, zoster universalis, zoster encephalitis and zoster osteoarthritis. Two of these forms, i.e., zoster

universalis and zoster osteoarthritis would appear to require special consideration.

It was formerly thought that herpes zoster and varicella were intimately related. Holt and McIntosh[22] state: "The view that herpes zoster is a manifestation of varicella no longer seems tenable." Dahl[23] (1946), in an extensive investigation, found no indication that the two diseases had a common etiology.

The zoster universalis that is described by some writers presumably might be confused with chickenpox since similar lesions have been described in poliomyelitis. In the New York epidemic of 1907 (Peabody, Draper and Dochez[24]), herpetic and vesicular eruptions were noted in a small percentage of cases of poliomyelitis. Brown[25] (1911) described a skin eruption in six consecutive cases of poliomyelitis. The lesions looked not unlike a small chicken pock, containing clear fluid. Batton[26] (1926) noted a rash which sometimes accompanies poliomyelitis. The lesions consisted of small vesicles widely distributed over the trunk and limbs. He points out that the vesicles resembled a small chicken pock.

The zona osteoarthritis that is mentioned above is suggestive of the arthritis that has been observed also in cases of poliomyelitis. McGugan[27] (1939) noted an unusually high incidence of joint involvement during an epidemic of poliomyelitis in Canada. Poynton[28] (1943), in England, found that arthritis is relatively common in some epidemics of poliomyelitis.

COMMENT

It is assumed, as a result of animal experiments, but not proven definitely, that a virus enters the human body to produce poliomyelitis. It has not been disproven that the so-called virus of poliomyelitis is not activated in the body of the affected individual as the result of an extrinsic factor or factors as in the case of herpes zoster.

The writer[29-34] (1946, 1947, 1948) has emphasized that the so-called virus of poliomyelitis is probably activated in the body as the result of the ingestion, during epidemics of poliomyelitis, of a toxic substance in food and water. The virus is excreted presumably from the body and does not, as commonly supposed, enter the body to cause disease. Proof of these views is indicated by three lines of investigation that have already been carried out.

On the assumption that the poliomyelitis virus enters the human body under natural circumstances through the nose and oropharynx as it does when placed in the nasal passages to produce disease in experimental animals, Schultz and Gebhardt[35, 36] (1937) found that when zinc sulfate was applied to the nasal mucosa of animals, they were protected from subsequent applications of the virus of poliomyelitis. As a result of this experiment, zinc sulfate was applied to the nostrils of thousands of children during an epidemic of poliomyelitis in Toronto in 1937 (Tisdall et al.[37]). The results of these experiments on humans, when analyzed, proved that zinc sulfate applications had been ineffective in protecting children from poliomyelitis during that epidemic as compared with results from the untreated control group.

Investigations by Faber and associates[38, 39] (1943, 1948) would indicate that the gastro-intestinal tract is likewise an improbable portal of entry of the virus of poliomyelitis into the human body. They showed that nontraumatic exposure of the stomach and intestine to poliomyelitis virus without simultaneous exposure of the oropharynx and upper esophagus failed to induce infection in a series of 44 cynomolgus monkeys. In a recent investigation (1948), Faber et al. gave large quantities of the virus as before, contained in capsules to avoid simultaneous contact of the oropharynx and upper esophagus, to 18 monkeys. In only one instance in these experiments was it possible to induce poliomyelitis on the 62 animals tested. In this one case it was thought possible that the virus had been regurgitated into the oropharynx. The writers concluded that the gastro-intestinal mucosa appears to be relatively impermeable to the poliomyelitis virus and may be regarded as an infrequent and exceptional portal of entry.

The skin as a portal of entry of the poliomyelitis virus, under natural circumstances, would presumably be affected in most instances only by a biting insect. Repeated experimental studies, however, indicate that an insect is probably not a factor during epidemics of poliomyelitis. In fact, wherever DDT has been employed on a vast scale to destroy insects in order to prevent the disease, the results have not been as expected and the disease has continued unabated.

Loveman[40] (1948) stated significantly: "Most of the conclusions concerning the method by which the virus gains access to the

human body have been drawn from experimental work done on rhesus monkeys, animals which actually are not susceptible to the disease. Many times these conclusions have not been correlated with clinical experience and simple logic; therefore, much chaos has arisen concerning the evaluation of these data." He also makes the following pertinent remark: "I have used the word 'virus' in the above statement, yet we cannot be certain that poliomyelitis actually is a viral disease."

Taylor[41] (1902) stated concerning poliomyelitis: "Its final place must be determined by a study of its cause or causes, as related to various other degenerations and inflamations of the nervous system." Jelliffe,[42] 30 years ago, cautioned: "The purity of laboratory experimentation is rarely repeated in nature."

There would appear to be only two possible lines of investigation in order to determine the pathogenesis of poliomyelitis, i.e., 1, (Exogenous) that a virus enters the human body under natural circumstances to cause the disease, and 2, (Endogenous) that a virus is activated within the human body as the result of an exogenous factor or factors, similar to the activation of the herpes zoster virus, and is subsequently excreted from the body. Every possible cause of poliomyelitis that has been suggested or that can be suggested can seemingly fall into one of these two categories for investigation. The exogenous origin of the virus of poliomyelitis has been intensively investigated for over forty years; the endogenous origin of the virus has been more or less ignored.

SUMMARY

1. It has been observed that epidemics of herpes zoster (acute posterior poliomyelitis) and acute anterior poliomyelitis occur in the same regions and in the same season of the year.

2. Herpes zoster is analogous to poliomyelitis in its precipitating factors, symptomatology and pathology. Flaccid paralysis may precede, accompany, or follow an attack of herpes zoster. Meningeal signs, with increased lymphocyte count and protein content in the spinal fluid, may occur in herpes zoster as in poliomyelitis.

3. Herpes zoster can result from extrinsic poisons, nonspecific bacterial toxins, and pressure on the spinal cord. Although herpes zoster is considered to be a virus disease, the virus is presumably activated in the human body as the result of the extrinsic poisons,

etc. It is pointed out that a similar situation probably exists in poliomyelitis, i.e., that the virus of this disease is activated in the human body as a result of a toxic substance that exists in food and water at the time and in the districts where epidemics of this disease are prevalent.

The writer wishes to express his appreciation to his wife, Alice Gilmore Scobey, R.N., for her helpful suggestions and cooperation in the preparation of this paper.

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CLINICAL REVIEW

In order to encourage the writing of clinical articles by recent graduates or senior medical students, the ARCHIVES *will publish monthly at least one such paper from the classes of Doctor Reuel A. Benson, New York Medical College, New York; Doctor Philip Moen Stimson, Cornell Medical School, New York, and Doctor John A. Toomey, Western Reserve University Medical School, Cleveland. Other interested medical schools are cordially invited to submit student papers for consideration.*

AMYOTONIA CONGENITA *

REPORT OF TWO CASES AND DISCUSSION

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The condition, amyotonia congenita, has many variations and different phases. In the two case reports which follow this fact can be clearly demonstrated even though a complete follow-up is lacking in one. The second case is merely presented to delineate these variations and indicate the prognostic difficulties of the condition. In the past ten years only two cases of the disease have been admitted to Metropolitan Hospital, New York, N. Y.

Amyotonia congenita was first described by Oppenheim in 1900; his name often designates the disease. Amyotonia is best described by a typical case history.

CASE REPORTS

Case 1. C. M., a Puerto Rican female, was born in Metropolitan Hospital, New York City, May 13, 1943. Her birth weight was five pounds twelve and one-half ounces. The mother had reached her twenty-ninth birthday and maintained a two year age superiority over her husband. The child was the result of a second pregnancy, but the first live-birth. The first pregnancy had been terminated by a spontaneous abortion. The fetus, a cephalic presentation, in the left occipito-anterior position, required low forceps in order to overcome a perineal arrest due to a prolonged first stage. The mother insisted it had been a very difficult labor. Wassermann and Kahn tests performed after delivery proved to be negative.

*Submitted as partial fulfillment of the requirements of the course in Senior Pediatrics at the New York Medical College, Flower and Fifth Avenue Hospitals.

No abnormalities were noted on the chart of the infant, and following the usual postpartum period of seven days, the mother and child had been discharged in good condition.

The child was admitted to St. Agnes Hospital, March 18, 1946, approximately three years old, previous to being institutionalized, since the family claimed it could not bear the difficulty in caring for the infant. This hospital lacked the facilities for the care of the child since it was essentially an acute hospital.

During its stay the patient developed both chickenpox and scarlet fever. The patient was then returned home to await a bed in an institution for chronic patients. At home the child developed an upper respiratory condition and entered Babies' Hospital, New York City, August 22, 1946, where the diagnosis of bronchopneumonia was entertained. Sulfa therapy was instituted and the child was transferred as unimproved to Metropolitan Hospital, September 10, 1946.

History. The mother had noticed that at nine months of age, when most babies sit up and crawl, her baby had difficulty in moving her legs. At that time the baby was taken to Babies' Hospital, but no prolonged therapy was considered. There was no family history of the disease. Upon searching the family background it was found that the mother resented the child since she had been conceived during the first month of marriage, and thus had deprived the mother of "much happiness". Then, too, following the discovery of the disease, the mother declined to care for the child in any way. It was inconvenient for her in every respect. The mother was a very nervous woman, who became tense and more irate with each question.

Physical Examination. Child was a 3½-year-old female having a skull which appeared to be hydrocephalic, lax extremities, and apparently no muscular tone. The child was unable to sit up. The trunk was poorly developed; the muscles of the arms showed no atrophy, and slight voluntary movement of poor quality was possible. The physiological reflexes were absent. The legs were moved easily, passively. The thighs were rotated outwardly, the knees flexed, and the feet were plantar flexed and inverted. There was complete lack of muscular tonicity and voluntary movement. The posterior muscle groups exhibited slight contractures. The ankle and knee jerk were absent, but the Babinski and Chaddock

gave varied responses with repeated attempts. This was ascribed to the possibility of voluntary movement of the toes upon stimulation of the foot. At no time could any loss of sensation of any kind be demonstrated.

The intelligence of the child appeared to be within normal limits, and, in many phases of the examination, exhibited responses far superior to the average child of three years.

Hospital Course. During the first seven months of hospitalization, the patient developed bronchopneumonia, measles and left lower lobe pneumonia. The lobar pneumonia persisted and intermittently a rise in temperature was noted associated with the typical signs of lobar pneumonia. Various examinations during these exacerbations indicated that the pneumonic process had spread to the opposite side .

An orthopedic consultation decided that casts could be applied to the legs to correct the contracture deformity, flexion of the knee. With regular physiotherapy, which consisted of passive exercises, heat and massage, some slight active motion was developed in both extremities. The upper extremity showed the more rapid improvement. It was still impossible to bear any weight, but the patient had developed some confidence and attempted with increased excitement to stand.

It had been decided that the casts would be applied when all indication of the pneumonic processes had been entirely eradicated. During the summer months of 1947 the patient was allowed all the sunshine and fresh air it was possible to have, continuing with the physiotherapy as outlined. Roentgenograms at this time, September 25, 1947, showed diffuse atrophy of bones, and a typical picture of healed rickets, especially that of the skull.

Before the casts could be applied the patient again developed lobar pneumonia, October 15, 1947. After a temporary clearing, following massive doses of penicillin and sulfa drugs, the left sided pneumonia had spread to the right chest. Streptomycin was added, 0.2 gms. per day. There was no response. On the tenth day following this bout of pneumonia, the temperature reached 106° F. In a few hours the child died.

Autopsy. Gross findings showed that the principal cause of death was bronchopneumonia with partial atelectasis of both lungs. Culture of the bronchial secretions were positive for Staphylo-

coccus aureus; N. Catarrhalis; Hem. Streptococcus; and Pneumococcus, Type 6.

Case 2. J. A., a Negro female child, was born May 19, 1935. She was the first live child of a mother who had previously had a stillbirth. The parturition period was of normal length and no birth trauma or abnormal delivery could be ascertained. The mother had received antiluetic therapy during the prenatal period.

History. At eight months it was noticed that the baby had not developed properly. She could not walk, sit up or even crawl. There had been no history of the condition in the families of either parent. It was learned that the parents had no desire for children, and that they were constantly inebriated, becoming very violent and aggressive.

The child had frequently suffered from "colds" and sore throats. The appetite had never been very good, and the child appeared lethargic. The baby had developed teeth at the proper time and now had the normal, or average number, for her age, fourteen.

Physical Examination. Patient was admitted December 26, 1936 with the diagnoses of amyotonia congenita, acute rhinopharyngitis and otitis media catarrhal acute. The child was a female Negro, 19 months of age, poorly nourished and poorly developed. Patient appeared flaccid, with the thighs rotated outwardly, knees flexed, and the feet plantar flexed and inverted.

Child was unable to sit up, there was a marked rachitic rosary, and Harrison's grooves were present. Also seen was frontal bossing. No epiphysitis could be demonstrated. The limbs were all hypotonic.

The pupils reacted to light and accommodation. There was no strabismus or nystagmus.

The abdomen was soft and protruded moderately.

The clitoris was hypertrophied, and there was marked hirsutism, including the presence of pubic hair. All of the physiological reflexes were absent. There was a definite bowing of the forearms, and a definite genu varus.

The intelligence of the child was not impaired and for her age had an excellent vocabulary and understood many reactions.

The Wassermann and Kahn tests were negative.

Hospital Course. During the first hospital admission of a month, all of the conditions present were cured with the exception of the

amyotonia. The child was able to sit up but was unsteady. The patient was discharged for clinic follow-up. The only therapy instituted for the amyotonia had been glycine.

Within a month the child was readmitted with all the conditions of the previous admission (February 10, 1937). The additional finding was an acute tonsillitis. During this admission patient developed right lower lobe pneumonia, with diagnosis proven by roentgenology. Child was discharged as unimproved to a convalescent home.

In less than four weeks the patient was admitted for the third time (April 14, 1937). There had been no improvement of the amyotonia during this period. She was placed on thyroid extract and a strychnine tonic, and assigned to physiotherapy for daily treatments. Gastroenteritis and an upper respiratory infection delayed progress, but following their eradication a marked improvement took place. Within four months (August 1, 1937) the muscular tone had improved and a walking motion developed as the patient learned to navigate a kiddy-car. The child spoke well, and was improving daily. At twenty-eight months the baby used sentences of six and seven words frequently.

During the following week the child attempted to walk with support and fared very well. Pneumonia intervened (September 22, 1937) but cured quickly, being followed by numerous upper respiratory infections which delayed further treatment. Finally, in February of the following year, the child was placed in Bryant's traction, for the correction of the contracture causing flexion of the knees. The three pound weights were removed in a month (March 17, 1938.)

The results were excellent, but the prolonged traction had caused the development of pressure sores over the tendo achillis. These healed nicely with the application of wet soaks and balsam of Peru.

Chickenpox delayed persistent therapy. In a month (April 18, 1938) the child could sit erect, and could move her legs again. They were still straight and traction was being applied only when the child was put to bed. Muscular tone gradually improved. Glycine, 15 gms. t.i.d., caused no definite improvement. Physiotherapy, never terminated, began to show its effect, especially following the daily whirlpool treatment. The patient was now forty months old (August 30, 1938).

Child continued to gain strength. The force of hand grip increased, although there appeared to be slight atrophy of the muscles of the shoulder girdle.

The lower extremities, though still weak and somewhat flaccid, could support the weight of the child. It was believed the child could voluntarily allow her limbs to support her. She could walk well with support. The abdominal reflexes were now positive.

Although a moderately severe diarrhea, acute bronchitis and acute tonsillitis and a left lower lobe pneumonia caused some regression, during the next few months the diseases were cured, and before the arrival of April 1939 the child could walk very well. The weakness remained, but the reflexes of the upper extremity were present; those of the lower extremity were weak and more often absent.

The condition improved gradually and finally, before the end of 1939, the child walked alone very well and could play with other children. In the early part of 1940 (January 13) her parents signed a release against advice with the end result that no further information concerning the child could be discovered. At the time of release the child was almost five years old.

DISCUSSION

The discussion will be limited to the pathology of amyotonia congenita.

Approximately 200 cases of amyotonia congenita have been reported. The disease is not hereditary but frequently occurs in siblings, twins and triplets. The pathology of the condition is not fully understood and some believe the disease to be some form of poliomyelitis.

Muscular Pathology. The pathology and histology in the muscles represent degenerative changes secondary to defective inervation of the muscles.

The muscle fibers are hypertrophied, some are of normal size and consistency, but many are small or hypoplastic. This is caused by a retardation in the development and maturation of the muscle fibers. There is no atrophy, merely hypoplasia of a large portion of the fibers of certain voluntary muscles.

There are also varying percentages of embryonal muscle spindles.

These muscle spindles have preserved cross striations and easily demonstrated Krause's and Hensen's membranes. There is little replacement by fat and no considerable increase of the connective tissue. The motor end plates are easily demonstrated. These findings enable a diagnosis to be made by biopsy of involved muscular tissue.

Neural Pathology. The dominant lesions of amyotonia congenita are found in the spinal cord. The cells of the anterior horn are degenerated, that is, reduced in number, and those remaining are distorted and atrophic, diminished in size. This, too, can be termed hypoplasia.

The spinal motor ganglion cells are often heterotopic. Some observers have found a malformation in the migration of these cells.

The anterior roots are diminished in size, and poorly or not myelinated. There is sometimes a noticeable slenderness and deficient myelinization of the peripheral nerves.

There is no demyelinization of the tracts of the spinal cord and no evidence of involvement of the pyramidal cells of the cerebral cortex.

In many cases there has been reported optic atrophy, with many of the individuals having either convergent or divergent strabismus together with malformations of other parts of the body.

Developmental Pathology. Amyotonia congenita is either the result of defective development of the fertilized ovum or of an essential biologic deficiency in one or both of the gametes. Most recent observations have not provided any support for a maldevelopment of the central nervous system referable to any injury to an originally normal fertilized ovum. There is no critical information to support a pathogenic theory based on continued destruction of fully formed spinal ganglion cells during fetal development.

The following observations are facts which tend to disprove such a theory: (1) Significant gliosis of the spinal cord has not been observed; (2) Published findings of ganglion cell degeneration can be attributed to terminal secondary pathologic processes either in the mother or in the child; (3) Progressive myelin degeneration of the anterior spinal nerve roots has not been observed, therefore, the characteristic absence of myelin sheaths may be considered a failure of development of these structures. Even in

cases in which the disease progresses rapidly, no inflammatory reaction has been described in the central nervous system, in the muscles or in the peripheral nerves.

SUMMARY

Amyotonia congenita may be considered a focal retardation or cessation in the development of voluntary muscles resulting from a malformation of the central nervous system, almost exclusively involving the peripheral motor neurons. This malformation occurs most likely before the sixth month of intra-uterine life.

Since the condition is fully developed before birth, improvement in or elimination of the basic defect is not to be expected, even though the disease may be compatible with life for many years.

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LEUKOTOMY IN CHILDREN. (Semaine des Hôpitaux de Paris, 25: 113, Jan. 10, 1949). Klein and Tardieu performed leukotomy on 8 idiotic children between the ages of 2 and 15 years, 5 of them aged less than 7 years. Bilateral section of the white matter was carried out with the aid of Cushing's trocar, on a plane corresponding to the coronal suture. There were no untoward reactions. The children seemed to feel comfortable immediately after the intervention. They remained quietly in bed, agitation subsided and they took notice of objects. They also acquired some automatisms. Institutionalization was no longer necessary. The children could be left alone with their toys; they were able to go around safely in the apartment and they could eat at the table. These results warrant leukotomy in idiotic children with an intelligence quotient of 20 to 30 or less, with generalized cerebral lesions demonstrated by electroencephalography or with a normal encephalogram but with diffuse lesions such as microhydrocephalus demonstrated by ventriculography—i. e., in those cases in which any other method of treatment does not offer any hope.—*Journal A.M.A.*

PEDIATRICS HALF A CENTURY AGO

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

INFECTIONS OF THE NEWBORN*

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We shall limit ourselves in this report to a brief statement concerning infections of the newborn based mainly on our own observations. We do not aim at a definite classification of these conditions; it is rather our desire to condemn certain attempts at classification that have been made. For instance, we do not incline to accept as pathological entities such conditions as melena neonatorum, hemorrhagic disease of the newborn, Buhl's disease, Winckel's disease, hemophilia of the newborn, etc. According to our observations and in accordance with the findings in the literature, all the clinical manifestations described under these various headings may exist as evidences of any one of a number of different infections, the nature and severity of the symptoms depending upon the character and virulence of the infecting organism and the degree of individual resistance. In the light of our present knowledge, therefore, and for the purposes of simplicity, and to stimulate investigation, we would include all these conditions under the term—infections of the newborn. If any attempt at classification is to be made today, it must be on a purely bacteriological basis.

In order to give a more definite idea of the type of cases which we would include under this heading, we cite a few illustrative instances:—

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Case 1. A male infant, born at term at the Maternity Hospital of Philadelphia after a normal labor. He weighed 3,350 grams at birth. The cord separated without incident rather earlier than usual. The infant continued normal until the sixteenth day, when he developed a slight stomatitis. The temperature was normal. On the following day, a slight elevation of temperature and green stools were noted. By the next day, there was an almost constant flow of blood from the mucous membrane of the mouth, marked exhaustion, inability to nurse, and bright red blood was passed in the stools. During the succeeding night and the next day (the day of death and the nineteenth since birth), the infant emaciated rapidly and had a series of convulsions which lasted up to the time of death.

Autopsy. The essential features noted were a hemorrhagic effusion around the umbilicus, a hemorrhage into the wall of the umbilical cord, a hemorrhagic effusion into the peritoneum and intense congestion of the mesentery. The mesenteric lymph nodes were not enlarged and there were no ecchymoses. The spleen was normal. The ileum contained altered blood. Neither the intestinal tract nor the stomach showed congestion or ulceration. The esophagus was filled with blood-stained mucus. The liver was enlarged, soft, and its appearance on section was distinctly cloudy. The adrenals and kidneys were normal. The brain was edematous and its membranes very cloudy, but otherwise normal.

An extensive histological examination was made which may be summed up as follows:—There was a generalized parenchymatous degeneration of the organs, a sclerosis of the pancreas, an acute enteritis and a commencing cirrhosis of the liver. Cultures were made at the autopsy from the heart's blood, left lung, spleen, liver, peritoneum, brain, contents of the ileum and the bile. The staphylococcus pyogenes aureus was recovered from the heart's blood, spleen and brain; the bacillus aerogenes lactis was determined in the heart's blood, spleen, peritoneum and in plates made from the contents of the ileum. From the bile were recovered the bacillus aerogenes lactis and a typical growth of the bacillus pyocyaneus. From the liver tissue, the three microorganisms mentioned were obtained. All three microorganisms, when isolated, proved pathogenic guinea pigs.

Case 2. For the clinical notes of this case we are indebted to Dr.

Bolles, resident physician of the Maternity Hospital. The infant was born after a normal labor on January 10, 1902. The child was not asphyxiated; there was no history or evidence of syphilis in the mother. His temperature was slightly elevated from the second day; on the seventh day he developed a papular eruption on his face. His temperature rose suddenly with the appearance of the eruption to 104° F. He took nourishment well and had no gastro-intestinal disturbance. His temperature fluctuated between 101° and 104° F., from the eighth to the twelfth day. The facial eruption persisted, and a similar eruption developed over the neck, shoulders and flexor surface of the arms. The persistent fever was attributed to this eruption.

On the thirteenth day "the baby developed symptoms suggestive of pneumonia"—rapid respiration and a harsh vesicular murmur. The temperature became a little more elevated. A cotton jacket was applied.

On the fourteenth day, marked twitching of the muscles of the right eye and constant lateral rotation of the head were noted. There were no general convulsions. The child did not cry out. He nursed poorly and was somewhat cyanosed.

On the fifteenth day the temperature was less high, 102° F., and he seemed generally better.

On the sixteenth day, the day of death, the temperature rose to 106.8° F. at 8 A.M. The child vomited a large quantity of blackish material, which was evidently altered blood, and had several tarry stools. The face became much cyanosed; great restlessness ensued, and death occurred rather suddenly at midday.

Autopsy was made by one of us twenty hours after death. A papulopustular eruption was present on the face, neck, forehead, shoulders, arms and upper part of the chest, in some areas it was hemorrhagic, especially over the flexor surfaces of the arms. The umbilical stump was not entirely healed, and rather unhealthy in appearance.

Thorax. Both auricles were distended with liquid blood, the right more than the left. The veins of the heart were greatly distended. The lungs showed some congestion at their margins.

Abdomen. The umbilical arteries contained thrombi. The principal lesions were some congestion of the vessels of the mesentery and enlargement and congestion of its lymph nodes.

The stomach contained some blackish material having the appearance of altered blood. There was no congestion or erosion of its mucous membrane. The intestines contained nothing abnormal. The liver on section was yellowish in color and rather soft. The spleen was moderately congested. The kidneys were anomalous.

Brain. On opening the cranium, following the lines of the sutures, there was found, overlying the left hemisphere, a large mass of clotted blood, more marked, anteriorly. This extended into the substance of the brain causing some destruction of tissue. Overlying the right hemisphere there was a less marked hemorrhage. The superior longitudinal, the left lateral and the straight sinus and the vena magna Galeni contained firmly organized thrombi. The veins of the brain throughout were distended with blood, and some of the superior cerebral veins on the left side contained thrombi; both lateral ventricles were dilated and contained considerable blood, especially the left.

Cultures were made on blood serum from the thrombus in the umbilical artery and from the spleen. There was a moderate growth on each tube. Cultures from the heart's blood were contaminated. Agar slant transfer showed profuse growth after twenty-four hours; one showed a pure growth and the other an almost pure growth of a bacillus possessing practically the same cultural characteristics as the colon bacillus, although not absolutely typical.

Case 3. The clinical notes of this case were also prepared by Dr. Bolles. The infant was born on April 3, in the Maternity Hospital, after a labor with a prolonged first stage. It showed no abnormality, but was poorly nourished and small. It nursed well at first. On the fifth day there was some oozing of blood about the matrix of the nail of the index finger of the right hand. On the same day it became jaundiced. On the sixth day it had a hemorrhage from the mouth while nursing and profuse bleeding from the first finger and thumb of the right hand and later in the day from all the fingers, together with some bleeding from the umbilicus. The infant cried and moaned constantly, apparently from pain. The jaundice became more marked and the breast was refused. During the following night there was a severe hemorrhage from the vagina, the rectum and the umbilicus. During

the seventh day a subcutaneous hemorrhage appeared over the middle third of the right tibia. Two convulsions occurred during the following night. The hemorrhages continued the following day and it was noted that the napkins were "soaked through." Emaciation became rapid and the jaundice more intense. There were some hemorrhages from the corneal vessels. The infant had several convulsions during the day; it became rapidly weaker and died after an extraordinarily profuse hemorrhage from the navel at 11 P. M.

Autopsy was made by one of us eleven hours after death. The principal lesions were subcutaneous hemorrhages over the extensor surface of the left thumb, the anterior surface of the left wrist and the outer surface of the tibia.

Abdomen. The mesenteric lymph nodes were enlarged and congested and the mesenteric vessels were full.

The intestines and stomach contained, throughout their course, a tarry substance resembling altered blood. There was moderate inflammation of the mucosa. There were several small hemorrhagic areas in the large intestine but no ulceration and no enlargement of the follicles. The bladder contained some bloody fluid. The vaginal mucous membrane was pale and there was no evidence of hemorrhage into the uterus.

The liver was enlarged and yellowish on its surface and on section. The left kidney showed an extensive hemorrhagic area limited to the anterior surface. There was no hemorrhage into the pelvis of the left kidney. The hemorrhagic area referred to was limited to the cortex. There was slight congestion of the left suprarenal. The right kidney showed numerous hemorrhagic areas throughout the medulla and cortex. There was no hemorrhage into the pelvis. The right suprarenal was more congested than the left and there was a slight hemorrhage into the medullary portion. The spleen was moderately enlarged and much congested. There were no thrombi in the renal vessels.

Thorax. There were numerous small hemorrhagic infarcts scattered through both lungs. There was some clotted blood in the larger branches of the right bronchus. The thymus gland was larger than normal but not congested. There was a small amount of fluid in the pericardium which was unusually yellowish in color. There was nothing abnormal about the heart.

Brain. There was considerable congestion of the veins of the brain, especially over the right hemisphere. There was very little blood and no thrombi in the sinuses. There was no hemorrhage into the ventricles nor into the substance of the brain.

Whether or not the bloody urine was a hematuria or hemoglobinuria was not determined. The case bore a close resemblance to socalled Winckel's disease.

Cultures made from the heart's blood, the clot in the umbilical artery and from the spleen showed a pure growth of the staphylococcus aureus. The microscopic examination of the liver showed some fatty infiltration, together with some fatty degeneration of the cells. Microscopic examination of the long bones showed no evidence of syphilis.

Case 4. Baby Young. Born on April 7, 1903, in the Maternity Hospital, after a normal labor, lasting eleven hours. The previous and present health of the mother was good. The father was healthy. There was no history of syphilis.

The temperature was elevated to 102° F., at birth. The condition of the infant was not brought to our attention by the hospital resident until about twenty-four hours before death. The stools had been abnormal since birth. On the third day, it was noted that they were greenish in color and slimy. The child nursed poorly. On the fifth day a small cyst, containing clear fluid, was observed in the sublingual region. The cyst had increased in size by the following day and the character of the stools remained unchanged. On the morning of the seventh day (the day of death), the child developed nystagmus and marked twitching of the muscles of the mouth. There was a slight bloody discharge from the lower bowel. The diarrhea became more marked. There were no hemorrhages from any other part of the body. The child became increasingly cyanotic. The pupils contracted; the cornea became somewhat opaque. Small papules and pustules were noted on the neck and face. The child moaned constantly and refused to nurse. Respirations became increasingly difficult; almost persistent marked contractions of the muscles of the neck and lower jaw developed with intermittent general convulsions. Death occurred in the middle of the afternoon.

Autopsy was made by one of us eighteen hours after death. The chief points of interest were a papular and papulopustular eruption

on the forehead, cheeks, neck and shoulders. There were ecchymotic areas on the neck. No eruption elsewhere. The umbilical stump had an entirely healthy appearance.

Abdomen. The mesentery was congested. The lymph nodes were enlarged and deeply cyanosed. The stomach contained a moderate amount of altered blood. There was decided congestion of the mucosa in the pyloric and cardiac areas; a few hemorrhagic areas but no ulceration. There was moderate congestion in the lower part of the ileum and one or two Peyer's patches were congested. The most marked congestion, however, was in the colon. This extended throughout its entire length, being especially evident in the upper part of the sigmoid and in the descending colon. There was marked enlargement of the isolated follicles and many ecchymotic areas, but no naked eye evidences of ulceration. The spleen was enlarged and purplish on section. The liver was enlarged and mottled, yellowish-purple on the surface and on section. Both kidneys were congested, especially the right, the latter containing some hemorrhagic infarcts. The right suprarenal gland was congested and the left hemorrhagic. The bladder was empty and there was no congestion or hemorrhage into its mucous membrane.

Thorax. The bronchial and all the mediastinal lymph nodes were enlarged and deeply congested; some of them almost hemorrhagic. Both lungs were generally congested but crepitant throughout. Both auricles and the vena cava were distended with liquid blood.

Cranium. The amount of cerebral fluid was increased above the normal. There was considerable fullness of the vessels at the base of the brain and moderate fullness of the superior cerebral veins. The sinuses were empty.

Cultures were made from the heart's blood, spleen, umbilical cord and lungs. A pure culture of the staphylococcus pyogenes aureus was obtained from the heart's blood, umbilical cord and the lungs. Cultures from the spleen were negative. Guinea pigs were inoculated from a first subculture. The organism did not prove pathogenic, probably owing to the well-known rapid deterioration in the virulence of this particular microorganism.

Case 5. Baby Letters. Born at Preston Retreat, after a normal labor on February 17, 1903. The temperature was normal at birth.

It rose to 101° F. the following day and continued above normal until death. On the third day, the temperature became suddenly elevated to 105° F. The child developed rapid (60 per minute) labored respirations and cyanosis. The cyanosis varied in its degree of intensity from time to time. The pulse rate increased with the respirations. On the fifth day, the child had green, mucous, undigested stools. Up to this time, there had been no intestinal symptoms. There were no hemorrhages at any time and no convulsions or muscular twitchings. The cyanosis and rapid respirations increased persistently up to the time of death on the seventh day and led to the diagnosis of pneumonia.

Autopsy was made by one of us ten hours after death. There were no skin eruptions. The umbilicus was healthy in appearance.

Abdomen. The umbilical vessels contained clots. The mesenteric vessels were full. There was marked congestion of the omentum. The large intestine was quite empty; the upper part of the small intestine and the stomach contained a bluish substance. On opening the colon, the mucous membrane was found extremely congested and in some places hemorrhagic. The same conditions existed in the small intestine. The blackish substance in the upper part of the small intestine and in the stomach was altered blood. The mucous membrane of the stomach was less congested than that of the intestines. The spleen was normal, the liver congested.

Thorax. The left pleural cavity contained about a tablespoonful of clotted and liquid blood. There were no adhesions between the parietal and visceral pleurae. The left lung contained numerous large hemorrhagic infarcts. The right lung was almost completely atelectatic. There was one hemorrhage infarct at its base, posteriorly. The bronchial lymph nodes were hemorrhagic. The minute vessels of the visceral and parietal pleuræ were very full. The point of origin of the hemorrhage could not be determined. The thymus, heart and vessels were normal. Examination of the brain was not permitted.

Cultures were made on agar-agar slants and in litmus milk from the clots in the umbilical vessels, the liver, the spleen, the blood in the pleural cavity and the heart's blood. Bouillon cultures were made also from the heart's blood. All these cultures were negative except the bouillon culture from the heart blood. From

this was obtained a pure growth of the bacillus coli immobilis. This microorganism, injected subcutaneously and intraperitonially, proved pathogenic to guinea pigs, causing diffuse subcutaneous hemorrhagic infiltration, hemorrhagic peritonitis, pleuritis and pericarditis, and enlargement and congestion of the liver, spleen and suprarenals. The same microorganism was obtained in pure culture from all the organs and the serous cavities.

There is one other case in our series, which showed much the same lesions and in which the bacillus coli immobilis was isolated. It was also pathogenic to guinea pigs causing similar lesions.

Case 6. Baby Smart. Was born in the Maternity Hospital on the morning of April 17, 1903. The temperature was normal at birth and had fallen to 97° F., by the evening of the seventeenth. It became elevated to 101° F., on the morning of the second day and continued above normal during the remainder of life, fluctuating between 99° and 104° F. On the third day the stools became yellowish-green and loose, and contained mucus. The child lost weight persistently from birth, but aside from the persistent temperature and correspondingly increased pulse and respiration rate and the character of the stools, nothing else abnormal was noted until the evening of April 26 (ninth day), when dusky red spots developed on the lower anterior portion of the tibia and on the extensor surface of the forearms. These areas rapidly became indurated and extended with marked rapidity involving, by the following morning, both cheeks, the buttocks, the flexor surfaces of the feet, the extensor surfaces of the left arm, the fingers of the left hand, but not the thumb. The areas on the face were circular in character, extending from above downward, from the lower lid of each eye to the base of the lower jaw, and anteroposteriorly from the nose to the ear. All of these areas became indurated. The redness disappeared on pressure of the finger, to return again quickly, there was no pitting and the margins were distinctly limited. During the course of the day, the index finger of the left hand became bluish-black, and, by the following morning (April 28), all of the fingers, a portion of the extensor surface of the left hand and, in fact, all of the indurated areas, were gangrenous. There was no change in the character of the fever at this time. The child emaciated rapidly and, on the evening of the 28th, had a slight intestinal hemorrhage. The respirations became

labored, muscular twitchings developed on the 29th and after midday the infant developed convulsions, which persisted until death, on the morning of April 30 (the thirteenth day).

The areas of induration described, aside from their color, closely simulated sclerema neonatorum. The area of distribution was suggestive, but the failure to spread after a period of ten or twelve hours was not entirely characteristic. Blood cultures were made during life, prior to the development of the condition just described, from a vein in the left forearm. Later cultures were made from blood obtained from an incision made over one of the areas of induration. At the time of this incision, it was noted that the surface redness was very superficial; that the deeper tissues were distinctly whitish in character and had the appearance of hardened subcutaneous fat.

Autopsy. A post-morten examination was obtained, only after much arguing with the father, and was limited absolutely to an abdominal incision. This incision was, however, extended sufficiently to enable us to expose the heart and to obtain cultures from the heart's blood. The principal changes noted were a mottled yellowish-purple color of the liver, a normal spleen, moderate enlargement and congestion of the mesenteric lymph nodes, and small clots in the umbilical vessels; there was moderate congestion of the mucosa of the lower portion of the large intestine. There was some altered blood in the stomach but no inflammation of its mucosa. The small intestine was normal. The bronchial and the mediastinal lymph nodes were deeply congested. Cultures were made from the spleen, the lungs, the umbilical vessels and the heart's blood. In all of the cultures made during life and in the cultures made from the heart's blood, post-morten, there was found the staphylococcus aureus and a streptococcus.

Etiology. Hemorrhagic conditions in the newborn may, in certain instances, be due to some of the causes formerly held accountable for all such manifestations. Among these may be mentioned birth trauma, fetal malformations, asphyxia and syphilis. In the vast majority of instances, however, hemorrhages may be considered as symptoms of an infectious condition.

Strong evidence in favor of the infectious nature of all such conditions, as melena, Winckel's disease, etc., rests in the fact that they are observed almost exclusively in institutions and not

uncommonly in epidemic form. One peculiar feature of this last observation is that bacteriological studies, while proving the cases infectious, have commonly failed to demonstrate the presence of the same microorganisms in all of the cases, a result which suggests that these infections are more often dependent upon the deficient technique of a poorly trained nurse than upon defective conditions in the ward.

The nature of the infecting organism varies widely. In the six cases we record, six different microorganisms were isolated, viz., the bacillus pyocyaneus, the bacillus lactis aerogenes, the colon bacillus, the staphylococcus aureus, the bacillus coli immobilis and a streptococcus. In our complete list of cases, amounting to about 15, only one other microorganism, an unclassified micrococcus, has been encountered. The literature, however, contains instances of infection by the pneumococcus, Pfeiffer's bacillus, the bacillus of Babés, the bacillus hemorrhagica of Kolb, the bacillus of Gaertner, the encapsulated bacillus of Dungern, the pneumococcus and a bacillus closely resembling the pneumococcus, but differing from it in certain essential characteristics (Kilham and Mercelis). Of these various microorganisms, the streptococcus, the bacillus coli commune and the staphylococcus are the ones which have been most commonly encountered. Delestre, for instance, in a series of ante- and post-mortem blood cultures made from infected infants, premature and born at term, obtained positive results in thirty-seven. In this number, the streptococcus was found fourteen times, the colon bacillus ten times and the staphylococcus six times.

Origin of These Infections. As has been said, these infections occur most frequently in maternity hospitals. The opportunities for the dissemination of disease germs in institutions are extraordinarily great. Without the utmost precaution, their presence is unavoidable. Bacteriological investigations have shown the presence of various microorganisms in the air and dust of the wards, especially in the region of the clothing from the patients and their beds. The air is probably infected through the medium of the clothing and hands of attendants, the stools and buccal secretions of the patients, the bed clothing and the infants' napkins. It is possible, therefore, that air infections do sometimes occur.

The mother's milk has been held accountable and doubtless

is responsible for a small percentage of cases. In the presence of suppurative lesions of the breast, pathogenic microorganisms are most liable to be introduced. In milk from apparently normal breasts, microorganisms, mainly the staphylococcus aureus and albus and the streptococcus, have been isolated. The nature of these microorganisms and the fact that in the udder of the cow concealed foci of suppuration are not uncommon, would suggest the likelihood of a similar condition in the human breast.

A source of infection that has been noted is the bath water of the infant. In certain instances, recorded in the literature, the use of the same water for the ablutions of several infants has resulted in infecting the water; bacteriological studies of such water have shown the presence of some of the microorganisms mentioned. The placing of infants in beds which have been previously occupied by infected infants and the bedding together of the infected and noninfected, has resulted in conveying the infection. Admitting, as we do, the accountability of all these sources, we are inclined to believe that the most common medium of infection is the poorly trained or careless nurse. In the institutions in which we have observed these cases, even when they have occurred in epidemic form, we have noted that the cases have not been confined to any one ward; that they have been handled by the same nurse and that the bacteriological studies have shown the presence of different microorganisms in the different cases. The records in the literature show very few instances of epidemics in which any one microorganism has been held accountable for all the cases. These facts, we think, argue against the greater frequency of air infections.

It is impossible to definitely fix upon the port of entry. The statements just made, together with our general observations, lead us to believe that the buccal cavity, the tonsils, pharynx and the remainder of the alimentary tract, are the most common. Next in order we would implicate the lungs. We believe that the cord has been given too great prominence as the point of entrance and that the other avenues named—the skin, conjunctiva, nose, ears and urogenital tract—are rarely responsible.

POST-MORTEM APPEARANCES. The appearances observed post-mortem may be summed up in the words "congestion" and "hemorrhage." These vary in great degree in different cases. In

some instances there are no hemorrhages whatsoever, but one rarely fails to find a considerable degree of congestion of the spleen, the mesenteric lymph nodes, the kidneys, suprarenals, liver, stomach or intestines. There is usually marked fullness of the mesenteric vessels; sometimes amounting to hemorrhage. The mucosa of the stomach and intestines, especially that of the large intestine, in many cases, is inflamed and thickened. The isolated follicles and Peyer's patches may or may not be enlarged. Ulceration in various portions of the gastrointestinal tract has been recorded. Ulceration of the duodenum just below the pylorus was observed by Landau and by him attributed to the blocking up of the gastroduodenal artery by an embolus, originating from a thrombus in the umbilical vein. Two similar cases have been reported by Kling. The liver, the kidneys and the suprarenal glands may be the seat of small or large hemorrhages. Instances have been recorded in which a hemorrhage into these organs or into the perirenal tissues has secondarily ruptured into the peritoneum, giving rise to profuse fatal hemorrhage. Bloody effusions into the serous sacks are of frequent occurrence. The bladder sometimes contains bloody urine, its mucous membrane may be inflamed and sometimes ecchymotic.

The changes commonly noted in the thoracic organs are: marked enlargement and congestion of the mediastinal nodes, various degrees of congestion in the lungs, hemorrhagic infarcts, limited areas of, or sometimes, complete atelectasis and occasionally pneumonia. The thymus is frequently congested, and may be hemorrhagic. The right auricle and ventricle ordinarily contain liquid or partially clotted blood. The heart muscle is sometimes soft and its vessels are frequently enormously distended. The blood in some infections seems to retain its fluidity to a remarkable degree, although instances of ante-mortem clotting have been noted. Thrombosis and embolism are common.

Brain. The changes consist of various degrees of thickening of the membranes, moderate or severe congestion, subdural hemorrhage, sometimes hemorrhage into the substance of the brain or into the ventricles. Extensive thrombosis of the sinuses and acute hydrocephalus have been noted.

Histological Studies have been comparatively few and incomplete. The principal changes are those commonly found in infec-

tious conditions, namely, cloudy swelling, fatty degeneration and infiltration of the liver and kidneys, in addition to the various changes resulting from hemorrhage.

SYMPTOMS. These infections may occur during, before or after labor. Most often they occur within the first week of life, although their onset may be delayed beyond the fifteenth day. Their degree of severity seems to differ according to the virulence of the infection. Profound toxic symptoms, suggesting disease of special organs, such as the lungs, may occur, the autopsy revealing absolutely no pulmonary lesion. On the other hand, the course of the infection may be attended by few symptoms. The symptoms have no definite order of occurrence. Fever is usually early manifested, and may vary from a slight elevation to hyperpyrexia, 108° F. having been recorded in one of our cases. Diarrhea, with malodorous, undigested, greenish, mucous stools, is present in the majority of cases. Icterus may occur and is sometimes intense. Skin eruptions of almost every description have been noted. The most common are: petechia, ecchymoses, bullæ, erythemata, erysipelatoid eruptions, and especially a papular or papulovesicular eruption, frequently going on to pustulation and involving the face, neck, shoulders and flexor surfaces of the arms. Apathy, rapid and persistent emaciation and inability or disinclination to nurse are always noted. Hemorrhage of varying degree, from either the skin, umbilical cord, eyes, ears, nose, mouth, vagina, bladder, stomach and bowels usually happens at some time during the course of the disease. Nervous phenomena are varied; they consist mainly of convulsions, retraction of the head, nystagmus, twitching of the muscles, tetanic and tonic spasms, strabismus, changes in the pupils, and sometimes paralysis. Cyanosis and rapid, labored and irregular respirations, usually toxic in origin, may occur early or, more frequently, late in the disease.

DIAGNOSIS. But little need be said in relation to the diagnosis of these conditions. Any decided elevation of temperature, notwithstanding the frequency of fever at this age, should arouse suspicion. It may be the only evidence of the condition for several days. In most of the cases we have seen, enteritis has been an early symptom, and next in order, perhaps, is the occurrence of the papular or papulovesicular eruption involving the skin of the face, neck, shoulders and forearms. If, therefore, one observes

this combination—fever, greenish, mucous stools, a skin eruption of the character described, and rapid emaciation—one is justified, from the therapeutic standpoint, in considering the condition infectious. If to these manifestations are added hemorrhage, nervous phenomena, cyanosis, and rapid, irregular or labored respiration, the evidences of an infectious condition are complete. Confirmatory data and definite information as to the character of the infection can be obtained by cultures made from the blood.

Several of the cases which we have had the privilege of studying only after death had been diagnosed inspiration pneumonia, this conclusion being based on the presence of fever, labored and rapid respiration, harsh respiratory murmur and cyanosis. Given the ordinarily accepted diagnostic symptoms of pneumonia in the newborn, rapid, labored respirations, recession of the chest wall and cyanosis, because of the comparative infrequency of pneumonia, the assumption that the condition is a blood infection rather than a true lung involvement, is justifiable. The differentiation between these conditions on the basis of the symptoms and physical signs, is not possible.

PROGNOSIS of these infections is distinctly bad. Mild infections do occur and, in some instances, the patients recover when the condition seems hopeless. It is a not uncommon occurrence, in private practice, to see mild cases of socalled melena recover, but in institutions the mortality from infections of any type is extraordinarily high.

PREVENTION. In the light of what has been said in reference to the source and mode of entrance of these infections, it is evident that their prevention is, in large part, possible, and that this is to be accomplished by measures directed toward the improvement of the general aseptic technique of our maternity hospitals. We would recommend that, in the construction of maternity hospitals, every effort be made to insure ease of cleanliness. Only the necessary attendants should be admitted to the wards. They should wear overgowns or large aprons and cuffs, which should be changed daily. Each morning before entering the wards their hands should be scrubbed for at least five minutes with soap and water, and washed again immediately after the changing of beds, the changing of napkins, or handling of the bed pan, and always before handling the infants. The general cleanliness of the nurse, especially the proper toilet of the mouth, should be insisted upon.

The infants should be kept in separate wards from the mothers and be in the charge of a special nurse. The resident and attending physicians should follow the same precautions suggested for the nurse.

We would recommend that the cord be tied, as is usual, before it is severed, with an aseptic ligature by aseptic hands, and that it be immediately dressed with a sterile dressing. We question, to some extent, the advisability of the routine measure of cleansing the infant's mouth, unless it is done under the most aseptic conditions.

The nurse who has handled the bedding of a patient, treated a fissured breast, emptied a bed pan or changed a napkin, unless she be most careful in preparing her hands, will readily infect the mouth of the infant. The condition of the breasts of the mother should be carefully studied. The recognition of erosions, fissures, or any other pathologic condition, should result in the immediate withdrawal of the infant. The common custom of suckling the infant under these conditions is to be strongly condemned. The normal breasts and surrounding areas should be most carefully cleansed before and after each nursing. Infants should not be permitted to suckle mothers suffering from puerperal septicemia. When cases of infection in the mother or infant occur, the wards which they have occupied should be closed and fumigated.

MEDICAL TREATMENT. Infection once established, there is comparatively little than can be done for these patients in the way of treatment. In an extensive epidemic of streptococcic infection, occurring in the Maternity Department of the University of Pennsylvania Hospital a few years ago, it was accidently discovered that two of the infants suffering from infection, when sent to their homes, rapidly recovered. It has been recorded, as a measure of treatment, that isolation of these cases, in hospitals, has a beneficial effect upon the patients. It is desirable, therefore, to isolate these infants as soon as suspicious symptoms arise, placing them in bright, sunny, well-ventilated rooms. Delestre, in making a series of blood cultures from infections of the newborn, under the direction of Hutinel, noted, to his great surprise, that the withdrawal of 2 or 3 ccm. of blood resulted in some improvement of the condition of the infant. Encouraged by this fact, he withdrew from some infants from 15 to 20 ccm. of blood, immediately injecting, subcutaneously, 20 to 30 ccm. of artificial

serum, with very beneficial results. He makes the following statement: "I have seen infants cyanosed, cold and practically moribund, resuscitated, positively, under my eyes as a result of this measure." He accounts for these results by the fact that the withdrawal of the blood acts in stimulating the activity of the hematopoietic and lymphopoietic organs.

We have had no experience with this measure. Judging from the difficulty one encounters in securing sufficient blood to make satisfactory blood cultures, one would naturally question the ability to secure this quantity of blood at a single bleeding, as Delestre claims to have done. In the light of the great mortality of these cases, and of the extravagant claim he makes for it, one would be justified in applying it.

The medicinal treatment is the same as that applied to all cases in which we are unable to influence the etiological factor, namely, the treatment of the symptoms. All infectious conditions, especially in those who are possessed of such low resistance, require vigorous stimulation and good nourishment. In some instances we have used high intestinal injections without beneficial results.

PNEUMONIA IN THE NEWBORN FROM INHALATION OF GASTRIC CONTENTS. (Archives of Disease in Childhood, London, 23: 254, Dec. 1948). Rhaney and Macgregor, in the course of performing postmortem examinations in cases of neonatal death, noted occurrence of pneumonia with certain special features, which they believe to be caused by the aspiration of stomach contents. An extensive survey of the available literature revealed that little attention had been devoted to this condition. The authors analyze clinical and necropsy data from 41 cases observed between 1939 and 1946. The main pathologic features were extensive consolidation with hemorrhage and a tendency to suppuration. Microscopically, digestion of tissues and lysis of blood in relation to aspirated material were characteristic. Attention is drawn to predisposing factors with particular reference to prematurity. There seems to have been an increase in the condition during the last two years under review, and it is assumed that this may be related to a shortage of the nursing staff.—*Journal A.M.A.*

BOOK REVIEWS

NEW GOULD MEDICAL DICTIONARY. Edited by Harold Wellington Jones, M.D., Normand L. Hoerr, M.D. and Arthur Osol, Ph.D. with the cooperation of an editorial board and eighty contributors. Pp. 1294 with 252 illustrations, 129 in color. Thin paper edition $10.75. Philadelphia: The Blakiston Company, 1949.

In reviewing a dictionary in any field or any language, it is practically impossible to give anything more than a passing impression of its worth. This has been true since the original scrofulous Sam Johnson penned his famous work. The importance of correct definition has been conceded for generations; certainly the legal profession expends considerable effort toward that goal. Furthermore, anyone interested in semantics appreciates the tremendous task involved in attempting succinct and authoritative information. The publishers should be congratulated on assembling an exceptionally fine staff in putting out this excellent volume. Apparently, it is the first non-revised medical dictionary to appear in a number of years. Derivation of words is clearly recorded, as for example antibiosis and antibiotic. The volume is academically sound and should prove a very material aid to medical students in their effort to separate the wheat from the chaff. In the opinion of this reviewer, this is an outstanding contribution to present day medicine and should prove of considerable value in allied fields. The work is enthusiastically recommended.

JOHN FITCH LANDON, M.D.

HEALTHY BABIES ARE HAPPY BABIES. By Josephine H. Kenyon, M.D. and Ruth K. Russell, M.D. Cloth. Pp. 310. Price $2.50. Boston: Atlantic-Little, Brown, 1949.

Drs. Kenyon and Russell, mother and daughter, collaborated nicely in writing this simple book for the prospective mother. The text is well written and the language has been simplified to make it quite intelligible for the average lay reader. The book makes no attempt to usurp the place of the doctor which is the common fault of most medical books aimed at the lay reader. The prospective mother and the parents of a newborn may read this book with benefit.

MICHAEL A. BRESCIA, M.D.

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